Although persistence of *B. melitensis* in wild ruminants has not been reported, and these animals are considered an epidemiologic dead-end reservoir (*3*), the unexpected prevalence observed (≈50%) suggests that Alpine ibex could be the source of bovine brucellosis reemergence in the study area in France. Strict surveillance policies have prevented infection of domestic livestock with *B. melitensis* in the study area since 1999. However, cohabitation of domestic and wild ruminants on pastures during the summer is rare but possible. Clinical signs and lesions observed in chamois and Alpine ibex are consistent with those reported for chamois and Alpine ibex with brucellosis (*4,5*). However, positive cultures were obtained from conventional target organs (knee, testes, and lymph nodes) but also from urogenital fluids, which indicates the potential for excretion of the organism.

The fact that births occur during periods and in places where female Alpine ibex are not in close contact with other wild/domestic species (because of higher altitude or rocky peaks) could explain the low transmission rate of *B. melitensis* to these animals. It also suggests that the venereal route might contribute to transmission within Alpine ibex during the mating season in winter. This report demonstrates the need for maintaining an active/reactive surveillance system for livestock and wildlife in brucellosis-free regions.

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Bruno Garin-Bastuji,[1] Jean Hars,[1] Antoine Drapeau, Moulay-Ali Cherfa, Yvette Game, Jean-Marie Le Horgne, Séverine Rautureau, Eric Maucci, Jean-Jacques Pasquier, Maryne Jay, and Virginie Mick

Author affiliations: French Agency for Food, Environmental, and Occupational Health and Safety, Maisons-Alfort, France (B. Garin-Bastuji, A. Drapeau, M.-A. Cherfa, M. Jay, V. Mick); French Hunting and Wildlife Agency, Gières, France (J. Hars); Departmental Veterinary Laboratory of Savoie, Chambery, France (Y. Game); Departmental Veterinary Services of Haute-Savoie, Chambery (J.-M. Le Horgne); French Ministry of Agriculture, Agro-Food Industry, and Forest, Paris, France (S. Rautureau); Departmental Veterinary Laboratory of Haute-Savoie, Seynod, France (E. Maucci); and Departmental Hunters Association of Haute-Savoie, Villy-le-Pelloux, France (J.-J. Pasquier)

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Address for correspondence: Bruno Garin-Bastuji, Laboratoire de Santé Animale, Unité Zoonoses Bactériennes, Agence Nationale de Sécurité Sanitaire de l'Alimentation, de l'Environnement et du Travail, 23 Avenue du Général-de-Gaulle, 94706 Maisons-Alfort Cedex, France; e-mail: bruno.garin-bastuji@anses.fr

[1]These authors contributed equally to this article.

Clostridium tetani Osteitis without Tetanus

To the Editor: Posttraumatic osteoarticular infections caused by *Clostridium* spp. are rare, and their outcomes are often unfavorable because of the persistence of the bacteria in bone (*1*,*2*). In a recent series of 12 patients (*2*), only 1 case of posttraumatic osteoarticular infection was caused by *C. tetani* (fracture of the distal humerus with polymicrobial infection). However, no information was available about the production of tetanospasmin by the infecting strain.

To the best of our knowledge, the only case of *C. tetani* infection with a toxigenic strain but without tetanus or osteitis was a wound infection that quickly improved after administration of antitetanus vaccine, prophylactic immunoglobulins, flucloxacillin, and metronidazole (*3*). The absence of clinical signs of tetanus despite chronic *C. tetani* infection probably resulted from vaccine-induced immunity and the fact that the patient received a booster vaccination and prophylactic immunoglobulins as soon as *C. tetani* had been identified. Retrospective immunochromatic testing of the patient's serum seemed to confirm this hypothesis. We report a case of osteitis caused by *C. tetani* in which clinical signs of tetanus did not develop despite production of tetanospasmin by the infecting strain.

In August 2011, a 26-year-old man was admitted to Nord Hospital in Marseille, France, because of an open fracture of his left tibia and fibula, contaminated with soil. The patient had been vaccinated against tetanus in 1997 and worked in scraps recycling, He rapidly underwent osteosynthesis (locking plates). Despite receiving oral amoxicillin–clavulanate (1 g 2 times/day) for 7 days, he was readmitted 12 days later for fever and suppuration of the leg wound and underwent a second surgical debridement. A bone biopsy sample revealed *Enterococcus faecalis*, *Enterobacter cloacae*, and *C. tetani*. Identification of *C. tetani* was confirmed by 16S rRNA amplification and sequencing (99.8% identity to *C. tetani,* GenBank accession no. AE015927). The organism was susceptible to amoxicillin, rifampin, vancomycin, and metronidazole. Because antitetanus vaccine had not been administered at the time of his previous hospital admission, a dose of vaccine and prophylactic immunoglobulins were administered at this time. Treatment with intravenous imipenem (1 g 3 times/day) plus oral ciprofloxacin (500 mg 3 times/day) was initiated for 1 month, followed by oral amoxicillin–clavulanate (1 g 2 times/day) plus ciprofloxacin (500 mg 3 times/day) for 1 month and then oral amoxicillin (2 g 3 times/day) for 2 months.

In February 2012, because bone consolidation had not occurred, the patient underwent surgical revision to remove the locking plate, clean the wound, and insert an external fixator. Cultures of specimens collected during surgery were negative. Serologic qualitative immunochromatic test result was positive for *C. tetani*. The patient received intravenous vancomycin and imipenem (1 g 2 times/day each) for 1 month, followed by oral amoxicillin (3 g 2 times/day), rifampin (300 mg 3 times/day), and ciprofloxacin (500 mg 3 times/day) for 3 months.

In July 2012 (11 months after the accident), because of fistula persistence, the patient underwent ablation of a tibial sequestrum (Figure) and implantation of a temporary cement spacer containing gentamicin and vancomycin. The only bacterium isolated from a tibial biopsy sample was *C. tetani*.

The causative strain was referred to the Centre National de Référence des Bactéries Anaérobies et du Botulisme, Pasteur Institute, Paris, where presence of the *tetX* gene encoding the tetanus neurotoxin was confirmed. Oral treatment with clindamycin (2.4 g/day) for 4 months was prescribed. However, because of the unfavorable outcome despite multiple interventions and antimicrobial drug regimens, the left leg was amputated 17 months after the accident.

Figure. Radiograph of left leg of patient with *Clostridium tetani* infection, showing delayed bone consolidation 11 months after fracture.

The case reported here is remarkable because clinical tetanus did not develop despite the production of tetanospasmin by the infecting strain and because late relapse occurred despite adapted treatment. The persistence of infection might be explained by a questionable initial antimicrobial drug regimen but also by spore formation and/or poor diffusion of antimicrobial drugs, as suggested by the presence of necrotic tissues such as the bone sequestrum. However, surgical revision, notably the ablation of this defect, should have facilitated the recovery and decreased bacterial concentration. In the literature, 3 cases of relapsing *C. tetani* infections have been reported, but those patients had not received antitetanus vaccine and they did show signs of tetanus (*4*,*5*); 1 of these patients with mandible necrosis experienced relapse 8 months after discontinuation of metronidazole.

The pathogenesis of *C. tetani* has mainly been attributed to its toxin. Our report suggests that *C. tetani* can also cause focal infections, notably severe chronic osteitis after open fractures, especially because the anatoxin-based antitetanus vaccine does not prevent colonization and infection.

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Pierre-Yves Levy, Pierre-Edouard Fournier, Laurène Lotte, Matthieu Million, Philippe Brouqui, and Didier Raoult

Author affiliations: Centre Hospitalier Universitaire de La Timone, Marseille, France (P.-Y. Levy, P.-E. Fournier, L. Lotte, D. Raoult); Aix-Marseille Université, Marseille (P.Y. Levy, P.-E. Fournier, M. Million, P. Brouqui, D. Raoult); Marseille Teaching Hospitals, Marseille (P.-Y. Levy, P.-E. Fournier); and Hôpital Nord, Marseille (P. Brouqui)

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Address for correspondence: Didier Raoult, Faculté de Médecine, URMITE, UM63, IRD198, CNRS7278, INSERM1095, Aix-Marseille Université, 13005 Marseille, France; email: didier.raoult@gmail.com

Rate of Congenital Toxoplasmosis in Large Integrated Health Care Setting, California, USA, 1998–2012

To the Editor: Although congenital toxoplasmosis occurs throughout the United States, little information is available about the rates of diagnosed illness in most of the nation, including California. Infection usually occurs by ingestion of undercooked meat and unwashed fruits and vegetables or exposure to soil or water contaminated with cat feces. Congenital transmission can occur when a woman is infected with *Toxoplasma gondii* during, or just before, pregnancy. Approximately 91% of women of childbearing age in the United States are susceptible to *T. gondii* infection (*1*). The United States has a low prevalence of *T. gondii* infection compared with many areas of the world (*2*). Severe congenital toxoplasmosis can result in hydrocephalus, retinochoroiditis that affects vision, microcephalus, seizures, hepatosplenomegaly, icterus, psychomotor retardation, and other sequelae (*3*). Infants with congenital toxoplasmosis are most often asymptomatic at birth; however, when severe symptoms occur, they are usually recognized and the condition diagnosed by the time the child is 2 years of age (*3*).

Our goal was to determine the rate of clinically identified cases of congenital toxoplasmosis in children from birth to 2 years of age within the Northern California Kaiser Permanente Medical Care Program (KPNC) during a 15-year period. KPNC is a group health plan that provides care for >3.2 million residents of northern California. The KPNC membership represents ≈30% of the insured population in the region and is demographically similar to the residents of the counties served except that the very poor and very wealthy are underrepresented (*4*).

We studied live births and infants during 1998–2012, the most recent 15-year period for which records were available and considered complete. We identified potential cases from KPNC electronic medical record databases and confirmed them by reviewing electronic and paper records. The system documents outside services, identified by the corresponding diagnostic codes or laboratory test codes. Eligible case-patients were infants, defined as <24 months of age, at the time of meeting any potential case criterion. We identified all births in which ICD-9-CM diagnostic codes for the mother or the infant included the following: 130-130.9 (toxoplasmosis), 771.2a (a special KPNC subset code specifying toxoplasmosis), and those with the more general 771.2 (congenital infections specific to the perinatal period) code; for the latter, an external special test for toxoplasmosis was assessed). We also identified all infants for whom any toxoplasmosis laboratory test had been ordered that had ≥1 of 23 specific KPNC laboratory codes for related serologic and PCR tests. We considered clinically confirmed case-patients to be infants with positive *T. gondii* IgM and/or IgA tests at <6 months of age, persistent IgG at >12 months of age, PCR–positive results for *T. gondii*, or diagnosis and care of toxoplasmosis-related conditions. To calculate 95% confidence intervals for rates, we used the exact binomial method.

During the 15-year study period, there were 521,655 live births at KPNC facilities and 2,010 infants received ≥1 test for toxoplasmosis. Ten infants met the potential case criteria of diagnostic codes; no additional patients met any of the case criteria by age 2 years. After electronic and paper charts were reviewed, 2 cases of congenital toxoplasmosis were confirmed. One case was diagnosed in 2003, the other in 2011. Both case-patients were girls: 1 was of Hispanic ethnicity and the other was of